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Search Results -

Term	Documents
CD40.USPT.	678
CD40S	0
CD40L.USPT.	162
CD40LS.USPT.	2
LIGAND.USPT.	37004
LIGANDS.USPT.	30360
GP39.USPT.	103
GP39S	0
OOPHORITIS.USPT.	41
OOPHORITI	0
TREAT$	0
((CD40 OR CD40L OR CD40 ADJ LIGAND OR GP39) AND (TREAT$ OR THERAP$ OR PREVENT$ OR INHIBIT$ OR BLOCK$ OR SUPPRESS$ OR ANTAGONI$) SAME (OOPHORITIS OR THYROID$)).USPT.	136

There are more results than shown above. Click here to view the entire set.

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L3

Refine Search

Recall Text | Clear

Search History

DATE: Wednesday, August 28, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L3	(cd40 or cd40L or cd40 adj ligand or gp39) and (treat$ or therap$ or prevent$ or inhibit$ or block$ or suppress$ or antagoni$) same (oophoritis or thyroid$)	136	L3
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L2	(cd40 or cd40L or cd40 adj ligand or gp39) and (treat$ or therap$ or prevent$ or inhibit$ or block$ or suppress$ or antagoni$) same (oophoritis or thyroid$)	284	L2
L1	(cd40 or cd40L or cd40 adj ligand or gp39) and (oophoritis or thyroid$)	375	L1

END OF SEARCH HISTORY

05744564 BIOSIS NO.: 000084092971
THE EFFECT OF ANTISERA TO THYMOSIN ALPHA-1 ON THE COURSE OF AUTOIMMUNE OVARIAN DYSGENESIS IN NEONATALLY THYMECTOMIZED MICE
AUTHOR: DE ANGELO L; MICHAEL S D
AUTHOR ADDRESS: DEP. BIOL. SCI., STATE UNIVERSITY OF NEW YORK, BINGHAMTON, NY 13901, USA.
JOURNAL: J REPROD IMMUNOL 11 (1). 1987. 41-54. 1987
FULL JOURNAL NAME: Journal of Reproductive Immunology
CODEN: JRIMD
RECORD TYPE: Abstract
LANGUAGE: ENGLISH----

12/7/89 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05924858 89028712 PMID: 2902931
Prevention and reversal of experimental autoimmune thyroiditis (EAT) in mice by administration of anti-L3T4 monoclonal antibody at different stages of disease development.
Stull S J; Kyriakos M; Sharp G C; Braley-Mullen H
Department of Medicine, University of Missouri School of Medicine, Columbia 65212.
Cellular immunology (UNITED STATES) Nov 1988, 117 (1) p188-98, ISSN 0008-8749 Journal Code: 1246405
Contract/Grant No.: DK35527; DK; NIDDK; DK36180; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Experimental autoimmune thyroiditis (EAT) can be induced in CBA/J mice following the transfer of spleen cells from mouse thyroglobulin (MTg)-sensitized donors that have been activated in vitro with MTg. Since L3T4+ T cells are required to transfer EAT in this model, the present study was undertaken to assess the effectiveness of the anti-L3T4 monoclonal antibody (mAb) GK1.5 in preventing or arresting the development of EAT. Spleen cells from mice given mAb GK1.5 prior to sensitization with MTg and adjuvant could not transfer EAT to normal recipients and cells from these mice did not proliferate in vitro to MTg. Donor mice given GK1.5 before immunization did not develop anti-MTg autoantibody and recipients of cells from such mice also produced little anti-MTg. GK1.5 could also prevent the proliferation and activation of sensitized effector cell precursors when added to in vitro cultures. When a single injection of mAb GK1.5 was given to recipients of in vitro-activated spleen cells, EAT was reduced whether the mAb was given prior to cell transfer or as late as 19 days after cell transfer. Whereas the incidence and severity of EAT was consistently reduced by injecting recipient mice with GK1.5, the same mice generally had no reduction in anti-MTg autoantibody. Since EAT is consistently induced in control recipients by 14-19 days after cell transfer, the ability of mAb GK1.5 to inhibit EAT when injected 14 or 19 days after cell transfer indicates that a single injection of the mAb GK1.5 can cause reversal of the histopathologic lesions of EAT in mice. These studies further establish the important role of L3T4+ T cells in the pathogenesis of EAT in mice and also suggest that therapy with an appropriate mAb may be an effective treatment for certain autoimmune diseases even when the therapy is initiated late in the course of the disease.
Record Date Created: 19881214

12/7/55 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

04063477 EMBASE No: 1989232519
Depletion of L3T4sup + and Lyt-2sup + cells by rat monoclonal antibodies alters the development of adoptively transferred experimental autoimmune thyroiditis
Flynn J.C.; Conaway D.H.; Cobbold S.; Waldmann H.; Kong Y.M.

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s

Set     Items   Description
S1        124   (GP39 OR CD40 OR CD40L OR CD40(W)LIGAND) AND (OOPHORITIS OR
                THYROID?)
S2         66   RD S1 (unique items)
S3          3   S2 AND PY<1996
S4        193   OOPHORITIS AND (T(W)CELL? OR T(W)LYMPHOCYT?)
S5        100   RD S4 (unique items)
S6         59   S5 AND PY<1996
S7       2506   THYROIDITIS AND (T(W)CELL? OR T(W)LYMPHOCYT?)
S8       1709   S7 AND PY<1996
S9        303   S8 AND ANTIBOD?(10N)(T(W)CELL? OR T(W)LYMPHOCYT?)
S10       196   RD S9 (unique items)
S11       105   S10 AND (TREAT? OR THERAP? OR PREVENT? OR INHIBIT? OR SUPP-
                RESS? OR ANTAGONI?)(10N)(ANTIBOD? OR T(W)CELL? OR T(W)LYMPYOC-
                YT?)
S12       105   RD S11 (unique items)
?
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s

Set     Items   Description
S1        124   (GP39 OR CD40 OR CD40L OR CD40(W)LIGAND) AND (OOPHORITIS OR
                THYROID?)
S2         66   RD S1 (unique items)
S3          3   S2 AND PY<1996
S4        193   OOPHORITIS AND (T(W)CELL? OR T(W)LYMPHOCYT?)
S5        100   RD S4 (unique items)
S6         59   S5 AND PY<1996
S7       2506   THYROIDITIS AND (T(W)CELL? OR T(W)LYMPHOCYT?)
S8       1709   S7 AND PY<1996
S9        303   S8 AND ANTIBOD?(10N)(T(W)CELL? OR T(W)LYMPHOCYT?)
S10       196   RD S9 (unique items)
S11       105   S10 AND (TREAT? OR THERAP? OR PREVENT? OR INHIBIT? OR SUPP-
                RESS? OR ANTAGONI?)(10N)(ANTIBOD? OR T(W)CELL? OR T(W)LYMPYOC-
                YT?)
S12       105   RD S11 (unique items)
? begin 357
       28aug02 13:30:10 User208760 Session D2137.3
           $27.35    4.885 DialUnits File5
             $138.25  79 Type(s) in Format  3
              $85.75  49 Type(s) in Format  7
          $224.00  128 Types
   $251.35  Estimated cost File5
           $58.60    6.511 DialUnits File73
              $45.00  18 Type(s) in Format  3
              $62.50  25 Type(s) in Format  7
          $107.50  43 Types
   $166.10  Estimated cost File73
           $19.40    6.061 DialUnits File155
               $2.52  12 Type(s) in Format  3
               $5.25  25 Type(s) in Format  7
            $7.77  37 Types
    $27.17  Estimated cost File155
           $71.27    5.679 DialUnits File399
              $52.25  19 Type(s) in Format  3
              $16.50  6 Type(s) in Format  7
           $68.75  25 Types
   $140.02  Estimated cost File399
            OneSearch, 4 files, 23.135 DialUnits FileOS
     $6.71  TELNET
   $591.35  Estimated cost this search
   $591.73  Estimated total session cost  23.302 DialUnits

File 357:Derwent Biotech Res.  1982-2002/June W1
       (c) 2002 Thomson Derwent & ISI
*File 357: File enhancements now online. See HELP NEWS 357.
Alert feature enhanced for multiple files, etc. See HELP ALERT.

      Set  Items  Description
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? s (gp39 or cd40 or cd40L or cd40(w)ligand) and (oophoritis or thyroid?)
             16  GP39
            166  CD40
             41  CD40L
            166  CD40
           3794  LIGAND
             64  CD40(W)LIGAND
              1  OOPHORITIS
            563  THYROID?
      S1      5  (GP39 OR CD40 OR CD40L OR CD40(W)LIGAND) AND (OOPHORITIS
                 OR THYROID?)
? t s1/3/all
```

Department of Immunology and Microbiology, Wayne State University School of Medicine, Detroit, MI 48201 United States
Cellular Immunology (CELL. IMMUNOL.) (United States) 1989, 122/2 (377-390)
CODEN: CLIMB ISSN: 0008-8749
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

To delineate the contribution of L3T4sup + and Lyt-2sup + cells in the pathogenesis of experimental autoimmune thyroiditis (EAT), synergistic pairs of monoclonal antibodies (mAb) to the T cell subsets were used in conjunction with the adoptive transfer of mouse thyroglobulin (MTg)-activated cells from immunized mice. Initial experiments verified the important role of L3T4sup + cells in the transfer of EAT. Subsequent experiments pointed to the relative contribution of both L3T4sup + and lyt-2sup + cells, depending on the stage and extent of disease development. Treatment during disease with L3T4, but not Lyt-2, mAb alone significantly reduced thyroiditis. However, in situ analysis of the cellular infiltrate in thyroid sections revealed that, after treatment with mAb, the appropriate subset was eliminated without altering the amount of the other subset in the remaining lesion. In addition, treatment during severe thyroiditis following the transfer of MTg-activated lymph node cells showed that Lyt-2 mAb alone also reduced thyroid infiltration. When the recipients were pretreated with either pair of mAb before transfer, disease development was only moderately affected. We conclude that (i) donor L3T4sup + cells ar the primary cells responsible for the initial transfer and development of thyroiditis; and (ii) previous in vitro cytotoxicity data, plus current monoclonal antibody treatment of disease and in situ analysis, further implicate a role for Lyt-2sup + cells in EAT pathogenesis.

7/53 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

04831706 EMBASE No: 1991326442
Suppression in murine experimental autoimmune thyroiditis: In vivo inhibition of CD4sup + T cell-mediated resistance by a nondepleting rat CD4 monoclonal antibody
Nabozny G.H.; Cobbold S.P.; Waldmann H.; Kong Y.-C.M.
Department of Immunology and Microbiology, Wayne State University School of Medicine, Detroit, MI United States
Cellular Immunology (CELL. IMMUNOL.) (United States) 1991, 138/1 (185-196)
CODEN: CLIMB ISSN: 0008-8749
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Genetically susceptible mice become resistant to experimental autoimmune thyroiditis (EAT) induction with mouse thyroglobulin (MTg) and lipopolysaccharide after pretreatment with deaggregated MTg (dMTg). Recent work showed this suppression to be mediated by CD4sup + suppressor T cells (T(s)). To study T(s) action in vivo, we used a rat IgG(2a) monoclonal antibody (mAb), YTS 177.9, which modulates CD4 antigen in vivo without depleting CD4sup + cells. Initial studies showed that after two 1-mg doses of mAb 7 days apart, extensive CD4 antigen modulation of peripheral blood leukocytes occurred within 4 days. Mice given CD4 mAb 24 hr before dMTg (2 doses, 7 days apart) were resistant to EAT induction when immunized with MTg and LPS 20 days later. Also, anti-rat IgG(2a) titers were reduced following challenge with heat-aggregated rat IgG(2a) compared to controls. Subsequent analysis of serum in CD4 mAb-treated animals revealed that mAb was present in the circulation for 14 days. Moreover, mice given CD4 mAb and dMTg, then challenged after only 10 days, when CD4 mAb was still circulating, developed a significantly higher incidence of thyroid damage than controls. These findings suggest that modulation of CD4 antigen does not interfere with T(s) activation, but the presence of CD4 mAb, at the time of autoantigenic challenge, can interfere with tolerance to EAT induction. Thus, the direct relationship between the presence of CD4 mAb and inhibition of EAT suppression implicates a role for

05744564 BIOSIS NO.: 000084092971
THE EFFECT OF ANTISERA TO THYMOSIN ALPHA-1 ON THE COURSE OF AUTOIMMUNE OVARIAN DYSGENESIS IN NEONATALLY THYMECTOMIZED MICE
AUTHOR: DE ANGELO L; MICHAEL S D
AUTHOR ADDRESS: DEP. BIOL. SCI., STATE UNIVERSITY OF NEW YORK, BINGHAMTON, NY 13901, USA.
JOURNAL: J REPROD IMMUNOL 11 (1). 1987. 41-54. 1987
FULL JOURNAL NAME: Journal of Reproductive Immunology
CODEN: JRIMD
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

12/7/89 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05924858 89028712 PMID: 2902931
Prevention and reversal of experimental autoimmune thyroiditis (EAT) in mice by administration of anti-L3T4 monoclonal antibody at different stages of disease development.
Stull S J; Kyriakos M; Sharp G C; Braley-Mullen H
Department of Medicine, University of Missouri School of Medicine, Columbia 65212.
Cellular immunology (UNITED STATES) Nov 1988, 117 (1) p188-98, ISSN 0008-8749 Journal Code: 1246405
Contract/Grant No.: DK35527; DK; NIDDK; DK36180; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Experimental autoimmune thyroiditis (EAT) can be induced in CBA/J mice following the transfer of spleen cells from mouse thyroglobulin (MTg)-sensitized donors that have been activated in vitro with MTg. Since L3T4+ T cells are required to transfer EAT in this model, the present study was undertaken to assess the effectiveness of the anti-L3T4 monoclonal antibody (mAb) GK1.5 in preventing or arresting the development of EAT. Spleen cells from mice given mAb GK1.5 prior to sensitization with MTg and adjuvant could not transfer EAT to normal recipients and cells from these mice did not proliferate in vitro to MTg. Donor mice given GK1.5 before immunization did not develop anti-MTg autoantibody and recipients of cells from such mice also produced little anti-MTg. GK1.5 could also prevent the proliferation and activation of sensitized effector cell precursors when added to in vitro cultures. When a single injection of mAb GK1.5 was given to recipients of in vitro-activated spleen cells, EAT was reduced whether the mAb was given prior to cell transfer or as late as 19 days after cell transfer. Whereas the incidence and severity of EAT was consistently reduced by injecting recipient mice with GK1.5, the same mice generally had no reduction in anti-MTg autoantibody. Since EAT is consistently induced in control recipients by 14-19 days after cell transfer, the ability of mAb GK1.5 to inhibit EAT when injected 14 or 19 days after cell transfer indicates that a single injection of the mAb GK1.5 can cause reversal of the histopathologic lesions of EAT in mice. These studies further establish the important role of L3T4+ T cells in the pathogenesis of EAT in mice and also suggest that therapy with an appropriate mAb may be an effective treatment for certain autoimmune diseases even when the therapy is initiated late in the course of the disease.
Record Date Created: 19881214

12/7/55 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

04063477 EMBASE No: 1989232519
Depletion of L3T4sup + and Lyt-2sup + cells by rat monoclonal antibodies alters the development of adoptively transferred experimental autoimmune thyroiditis
Flynn J.C.; Conaway D.H.; Cobbold S.; Waldmann H.; Kong Y.M.

1/3/1
DIALOG(R)File 357:Derwent Biotech Res.
(c) 2002 Thomson Derwent & ISI. All rts. reserv.

0281123 DBA Accession No.: 2001-03467 PATENT
A monoantigen-presenting cell capable of dividing and with suppressed functionality of the co-stimulatory receptors B7 and **CD40** - virus, bacterium or plasmid vector-mediated DNA, RNA or ribozyme gene transfer, expression in monocyte, dendritic cell or macrophage for autoimmune disease gene therapy
AUTHOR: Sheriff A; Gebauer F
CORPORATE SOURCE: Berlin, Germany.
PATENT ASSIGNEE: Sheriff A 2000
PATENT NUMBER: WO 200066715 PATENT DATE: 20001109 WPI ACCESSION NO.: 2001-015978 (200102)
PRIORITY APPLIC. NO.: DE 1044858 APPLIC. DATE: 19990918
NATIONAL APPLIC. NO.: WO 2000EP3984 APPLIC. DATE: 20000504
LANGUAGE: German

1/3/2
DIALOG(R)File 357:Derwent Biotech Res.
(c) 2002 Thomson Derwent & ISI. All rts. reserv.

0269745 DBA Accession No.: 2001-09499 PATENT
Composition useful for treating autoimmune disease such as psoriasis, Hashimoto's **thyroiditis**, lupus, rheumatoid arthritis, multiple sclerosis and neoplastic diseases, comprising a **CD40** antagonist - **CD40**-antagonist, humanized antibody and monoclonal antibody
AUTHOR: Chu K; Wang C
CORPORATE SOURCE: Emeryville, CA, USA.
PATENT ASSIGNEE: Chiron 2001
PATENT NUMBER: WO 200124823 PATENT DATE: 20010412 WPI ACCESSION NO.: 2001-281719 (2029)
PRIORITY APPLIC. NO.: US 157461 APPLIC. DATE: 19991004
NATIONAL APPLIC. NO.: WO 2000US27184 APPLIC. DATE: 20001002
LANGUAGE: English

1/3/3
DIALOG(R)File 357:Derwent Biotech Res.
(c) 2002 Thomson Derwent & ISI. All rts. reserv.

0233339 DBA Accession No.: 99-03440 PATENT
Treatment of autoimmune disease using agent that blocks interaction of CD34 with CD154 - humanized antibody monoclonal antibody used to treat e.g. dermatitis, lupus erythematosus, diabetes, multiple sclerosis, optionally via a gene therapy vector
AUTHOR: Thomas D W
CORPORATE SOURCE: Cambridge, MA, USA.
PATENT ASSIGNEE: Biogen 1999
PATENT NUMBER: WO 9900143 PATENT DATE: 990107 WPI ACCESSION NO.: 99-095475 (9908)
PRIORITY APPLIC. NO.: US 51484 APPLIC. DATE: 970701
NATIONAL APPLIC. NO.: WO 98US13284 APPLIC. DATE: 980626
LANGUAGE: English

1/3/4
DIALOG(R)File 357:Derwent Biotech Res.
(c) 2002 Thomson Derwent & ISI. All rts. reserv.

0213000 DBA Accession No.: 97-08121 PATENT

Humanized antibody capable of competing with, or derived from, murine 24-31 antibody - antibody engineering for use as an immunosuppressive in autoimmune disease therapy
AUTHOR: Black A; Hanna N; Padlan E A; Newman R A
CORPORATE SOURCE: San Diego, CA, USA.
PATENT ASSIGNEE: Idec-Pharm. 1997
PATENT NUMBER: WO 9717446 PATENT DATE: 970515 WPI ACCESSION NO.: 97-281035 (9725)
PRIORITY APPLIC. NO.: US US554840 APPLIC. DATE: 951107
NATIONAL APPLIC. NO.: WO 96US17875 APPLIC. DATE: 961107
LANGUAGE: English

1/3/5
DIALOG(R)File 357:Derwent Biotech Res.

0208642 DBA Accession No.: 97-03763 PATENT
Treating or preventing T-cell-mediated disorder with antibody to T-cell surface receptor - e.g. multiple sclerosis therapy using an immunosuppressive monoclonal antibody, chimeric antibody or humanized antibody
AUTHOR: Noelle R J; Claassen E
CORPORATE SOURCE: Hanover, NH, USA; Delft, The Netherlands.
PATENT ASSIGNEE: Dartmouth-Coll.; TNO 1996
PATENT NUMBER: WO 9640246 PATENT DATE: 961219 WPI ACCESSION NO.: 97-108629 (9710)
PRIORITY APPLIC. NO.: US 481735 APPLIC. DATE: 950607
NATIONAL APPLIC. NO.: WO 96US9137 APPLIC. DATE: 960606
LANGUAGE: English
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Search Results -

Term	Documents
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CD40LS.USPT.	2
LIGAND.USPT.	37004
LIGANDS.USPT.	30360
GP39.USPT.	103
GP39S	0
OOPHORITIS.USPT.	41
OOPHORITI	0
TREAT$	0
((CD40 OR CD40L OR CD40 ADJ LIGAND OR GP39) AND (TREAT$ OR THERAP$ OR PREVENT$ OR INHIBIT$ OR BLOCK$ OR SUPPRESS$ OR ANTAGONI$) SAME (OOPHORITIS OR THYROID$)).USPT.	136

There are more results than shown above. Click here to view the entire set.

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L3

Refine Search

Recall Text | Clear

Search History

DATE: Wednesday, August 28, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L3	(cd40 or cd40L or cd40 adj ligand or gp39) and (treat$ or therap$ or prevent$ or inhibit$ or block$ or suppress$ or antagoni$) same (oophoritis or thyroid$)	136	L3
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L2	(cd40 or cd40L or cd40 adj ligand or gp39) and (treat$ or therap$ or prevent$ or inhibit$ or block$ or suppress$ or antagoni$) same (oophoritis or thyroid$)	284	L2
L1	(cd40 or cd40L or cd40 adj ligand or gp39) and (oophoritis or thyroid$)	375	L1

END OF SEARCH HISTORY

Gambel, Phillip

From: Gambel, Phillip
Sent: Wednesday, August 28, 2002 1:19 PM
T : 'garcia@cua.edu'
Subject: FW: request for class / section change for phillip gambel

in case you did not get the first email, i am sending it again

---

Dean Garcia,

I was not on the class list for

Criminal Law 275
Section 01
Fall 2002
Professor Leroy Clark.

Professor Clark indicated that you should be contacted to update the class
list.

Could you please remedy this in view of the email below.

Thanx.

Phillip Gambel
Student Number 2043524

-----Original Message-----
From: GORMLEY@law.edu [mailto:GORMLEY@law.edu]
Sent: Wednesday, August 14, 2002 4:07 PM
To: Phillip.Gambel@USPTO.GOV
Cc: NIEDZIELKO@law.edu
Subject: request for class / section change for phillip gambel

Phillip,

I received your request from Dean Niedzielko and have registered you in Criminal Law (275 -Section 1). Please let me know if you have any further questions or concerns.

Regards,

Laura Gormley
Columbus School of Law
Registrar/Director of Academic Services
(202) 319-5003

From: Phillip.Gambel@USPTO.GOV [mailto:Phillip.Gambel@USPTO.GOV]
Sent: Tuesday, August 06, 2002 4:19 PM
To: NIEDZIELKO@law.edu
Subject: request for class / section change for phillip gambel

Dean Niedzielko
I am about to start my second year of evening law classes and I would like to request a change in the Section for Criminal Law 275.
I am currently enrolled in Law 275 / Criminal Law / Class Number 3463 / Section 2.
However, I would prefer to be enrolled in Law 275 Criminaal Law / Class

Number 1489 / Section 01.
My study partner from the first year is in Section 01 and I would prefer if we could stay in the same Section in Criminal Law.
We are in the same Courses / Sections for other classes.
Please let me know if it is possible to change Sections and how I go about doing so.
Thanx for your time and attention
Phillip Gambel
703-308-3997

Gambel, Phillip

From: Gambel, Phillip
Sent: Wednesday, August 28, 2002 2:40 PM
To: STIC-ILL
Subject: noell e amendment 09/2236334

stic

please provide the following references to

phillip gambel
art unit 1644
308-3997

1644 mailbox 9E12

10172972 BIOSIS NO.: 199698627890
Functional interactions of T cells with endothelial cells: The role of CD40L-CD40-mediated signals.
AUTHOR: Yellin Michael J(a); Brett Jerald; Baum David; Matsushima Anne; Szabolcs Matthias; Stern David; Chess Leonard
AUTHOR ADDRESS: (a)Columbia Univ., Div. Rheumatol., Black Build., 8-808, 630 W. 168 St., New York, NY 10032**USA
JOURNAL: Journal of Experimental Medicine 182 (6):p1857-1864 1995
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

PLEASE PROVIDE COPY OF THE FRONT OF THE JOURNAL. THANX.

3/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

09885688 BIOSIS NO.: 199598340606
Contribution of CD28/CTLA4/B7 and gp39/CD40 costimulation pathways in clonal expansion and functional acquisition of self reactive T cells.
AUTHOR: Griggs Nathan(a); Agersborg Sally; Noelle Randolf; Ledbetter Jeffrey; Linsley Peter; Tung Kenneth
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Virginia, Charlottesville, VA 22908 **USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (21A):p141 1995
CONFERENCE/MEETING: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

3/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

09410720 BIOSIS NO.: 199497419090
Human monoclonal autoantibodies specific for the bullous pemphigoid antigen 1 (BPAg 1).
AUTHOR: Peyron Eric; Nicolas Jean-Francois; Reano Alain; Roche Pascale; Thivolet Jean; Haftek Marek; Schmitt Daniel; Peronne Catherine; Banchereau Jacques; Rousset Francoise
AUTHOR ADDRESS: INSERM U346, Hopital Edouard Herriot, 69467 Lyon Cedex 03 **France
JOURNAL: Journal of Immunology 153 (3):p1333-1339 1994
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/45 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2002 Elsevier Science B.V. All rts. reserv.

04764451 EMBASE No: 1991257805
Experimental autoimmune oophoritis. II. Both lymphoid cells and antibodies are successful in adoptive transfer
Damjanovic M.
Immunology Research Center, Vojvode Stepe 458, 11221 Belgrade Yugoslavia
Autoimmunity (AUTOIMMUNITY) (United Kingdom) 1991, 9/3 (217-223)
CODEN: AUIME ISSN: 0891-6934
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH

6/3/46 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2002 Elsevier Science B.V. All rts. reserv.

04216077 EMBASE No: 1990098619
Autoimmune orchitis and oophoritis
Tung K.S.K.; Mahi-Brown C.A.
Washington University School of Medicine, Box 8118, 660 South Euclid Avenue, St. Louis, MO 63110 United States
Immunology and Allergy Clinics of North America (IMMUNOL. ALLERGY CLIN. NORTH AM.) (United States) 1990, 10/1 (199-214+ix)
CODEN: INCAE ISSN: 0889-8561
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

6/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09885688 BIOSIS NO.: 199598340606
Contribution of CD28/CTLA4/B7 and gp39/CD40 costimulation pathways in clonal expansion and functional acquisition of self reactive T cells.
AUTHOR: Griggs Nathan(a); Agersborg Sally; Noelle Randolf; Ledbetter Jeffrey; Linsley Peter; Tung Kenneth
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Virginia, Charlottesville, VA 22908

**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (21A):p141
1995
CONFERENCE/MEETING: Keystone Symposium on Control and Manipulation of the
Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

6/3/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

05744564 BIOSIS NO.: 000084092971
THE EFFECT OF ANTISERA TO THYMOSIN ALPHA-1 ON THE COURSE OF AUTOIMMUNE
OVARIAN DYSGENESIS IN NEONATALLY THYMECTOMIZED MICE
AUTHOR: DE ANGELO L; MICHAEL S D
AUTHOR ADDRESS: DEP. BIOL. SCI., STATE UNIVERSITY OF NEW YORK, BINGHAMTON,
NY 13901, USA.
JOURNAL: J REPROD IMMUNOL 11 (1). 1987. 41-54. 1987
FULL JOURNAL NAME: Journal of Reproductive Immunology
CODEN: JRIMD
RECORD TYPE: Abstract
LANGUAGE: ENGLISH----

12/7/89 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05924858 89028712 PMID: 2902931
Prevention and reversal of experimental autoimmune thyroiditis
(EAT) in mice by administration of anti-L3T4 monoclonal antibody at
different stages of disease development.
Stull S J; Kyriakos M; Sharp G C; Braley-Mullen H
Department of Medicine, University of Missouri School of Medicine,
Columbia 65212.
Cellular immunology (UNITED STATES) Nov 1988, 117 (1) p188-98,
ISSN 0008-8749 Journal Code: 1246405
Contract/Grant No.: DK35527; DK; NIDDK; DK36180; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Experimental autoimmune thyroiditis (EAT) can be induced in CBA/J
mice following the transfer of spleen cells from mouse thyroglobulin
(MTg)-sensitized donors that have been activated in vitro with MTg. Since
L3T4+ T cells are required to transfer EAT in this model, the
present study was undertaken to assess the effectiveness of the anti-L3T4
monoclonal antibody (mAb) GK1.5 in preventing or arresting the
development of EAT. Spleen cells from mice given mAb GK1.5 prior to
sensitization with MTg and adjuvant could not transfer EAT to normal
recipients and cells from these mice did not proliferate in vitro to MTg.
Donor mice given GK1.5 before immunization did not develop anti-MTg
autoantibody and recipients of cells from such mice also produced little
anti-MTg. GK1.5 could also prevent the proliferation and activation of
sensitized effector cell precursors when added to in vitro cultures. When a
single injection of mAb GK1.5 was given to recipients of in vitro-activated
spleen cells, EAT was reduced whether the mAb was given prior to cell
transfer or as late as 19 days after cell transfer. Whereas the incidence
and severity of EAT was consistently reduced by injecting recipient mice
with GK1.5, the same mice generally had no reduction in anti-MTg

autoantibody. Since EAT is consistently induced in control recipients by 14-19 days after cell transfer, the ability of mAb GK1.5 to inhibit EAT when injected 14 or 19 days after cell transfer indicates that a single injection of the mAb GK1.5 can cause reversal of the histopathologic lesions of EAT in mice. These studies further establish the important role of L3T4+ T cells in the pathogenesis of EAT in mice and also suggest that therapy with an appropriate mAb may be an effective treatment for certain autoimmune diseases even when the therapy is initiated late in the course of the disease.
Record Date Created: 19881214

12/7/55 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

04063477 EMBASE No: 1989232519
Depletion of L3T4sup + and Lyt-2sup + cells by rat monoclonal antibodies alters the development of adoptively transferred experimental autoimmune thyroiditis
Flynn J.C.; Conaway D.H.; Cobbold S.; Waldmann H.; Kong Y.M.
Department of Immunology and Microbiology, Wayne State University School of Medicine, Detroit, MI 48201 United States
Cellular Immunology (CELL. IMMUNOL.) (United States) 1989, 122/2 (377-390)
CODEN: CLIMB ISSN: 0008-8749
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

To delineate the contribution of L3T4sup + and Lyt-2sup + cells in the pathogenesis of experimental autoimmune thyroiditis (EAT), synergistic pairs of monoclonal antibodies (mAb) to the T cell subsets were used in conjunction with the adoptive transfer of mouse thyroglobulin (MTg)-activated cells from immunized mice. Initial experiments verified the important role of L3T4sup + cells in the transfer of EAT. Subsequent experiments pointed to the relative contribution of both L3T4sup + and lyt-2sup + cells, depending on the stage and extent of disease development. Treatment during disease with L3T4, but not Lyt-2, mAb alone significantly reduced thyroiditis. However, in situ analysis of the cellular infiltrate in thyroid sections revealed that, after treatment with mAb, the appropriate subset was eliminated without altering the amount of the other subset in the remaining lesion. In addition, treatment during severe thyroiditis following the transfer of MTg-activated lymph node cells showed that Lyt-2 mAb alone also reduced thyroid infiltration. When the recipients were pretreated with either pair of mAb before transfer, disease development was only moderately affected. We conclude that (i) donor L3T4sup + cells ar the primary cells responsible for the initial transfer and development of thyroiditis; and (ii) previous in vitro cytotoxicity data, plus current monoclonal antibody treatment of disease and in situ analysis, further implicate a role for Lyt-2sup + cells in EAT pathogenesis.

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7/53 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

04831706 EMBASE No: 1991326442
Suppression in murine experimental autoimmune thyroiditis: In vivo inhibition of CD4sup + T cell-mediated resistance by a nondepleting rat CD4 monoclonal antibody
Nabozny G.H.; Cobbold S.P.; Waldmann H.; Kong Y.-C.M.
Department of Immunology and Microbiology, Wayne State University School

of Medicine, Detroit, MI United States
Cellular Immunology (CELL. IMMUNOL.) (United States) 1991, 138/1 (185-196)
CODEN: CLIMB ISSN: 0008-8749
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Genetically susceptible mice become resistant to experimental autoimmune thyroiditis (EAT) induction with mouse thyroglobulin (MTg) and lipopolysaccharide after pretreatment with deaggregated MTg (dMTg). Recent work showed this suppression to be mediated by CD4sup + suppressor T cells (T(s)). To study T(s) action in vivo, we used a rat IgG(2a) monoclonal antibody (mAb), YTS 177.9, which modulates CD4 antigen in vivo without depleting CD4sup + cells. Initial studies showed that after two 1-mg doses of mAb 7 days apart, extensive CD4 antigen modulation of peripheral blood leukocytes occurred within 4 days. Mice given CD4 mAb 24 hr before dMTg (2 doses, 7 days apart) were resistant to EAT induction when immunized with MTg and LPS 20 days later. Also, anti-rat IgG(2a) titers were reduced following challenge with heat-aggregated rat IgG(2a) compared to controls. Subsequent analysis of serum in CD4 mAb-treated animals revealed that mAb was present in the circulation for 14 days. Moreover, mice given CD4 mAb and dMTg, then challenged after only 10 days, when CD4 mAb was still circulating, developed a significantly higher incidence of thyroid damage than controls. These findings suggest that modulation of CD4 antigen does not interfere with T(s) activation, but the presence of CD4 mAb, at the time of autoantigenic challenge, can interfere with tolerance to EAT induction. Thus, the direct relationship between the presence of CD4 mAb and inhibition of EAT suppression implicates a role for CD4 molecules in the mediation of suppression.

07906301 BIOSIS NO.: 000093005424
SUPPRESSION IN MURINE EXPERIMENTAL AUTOIMMUNE THYROIDITIS IN-VIVO INHIBITION OF CD4-POSITIVE T CELL-MEDIATED RESISTANCE BY A NONDEPLETING RAT CD4 MONOCLONAL ANTIBODY
AUTHOR: NABOZNY G H; COBBOLD S P; WALDMANN H; KONG Y-C M
AUTHOR ADDRESS: DEP. IMMUNOL. MICROBIOL, WAYNE STATE UNIV. SCH. MED., DETROIT, MICH.
JOURNAL: CELL IMMUNOL 138 (1). 1991. 185-196. 1991
FULL JOURNAL NAME: Cellular Immunology
CODEN: CLIMB
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Genetically susceptible mice become resistant to experimental autoimmune thyroiditis (EAT) induction with mouse thyroglobulin (MTg) and lipopolysaccharide after pretreatment with deaggregated MTg (dMTg). Recent work showed this suppression to be mediated by CD4+ suppressor T cells (Ts). To study Ts action in vivo, we used a rat IgG2a monoclonal antibody (mAb), YTS 177.9, which modulates CD4 antigen in vivo without depleting CD4+ cells. Initial studies showed that after two 1-mg doses of mAb 7 days apart, extensive CD4 antigen modulation of peripheral blood leukocytes occurred within 4 days. Mice given CD4 mAb 24 hr before dMTg (2 doses, 7 days apart) were resistant to EAT induction when immunized with MTg and LPS 20 days later. Also, anti-rat IgG2a titers were reduced following challenge with heat-aggregated rat IgG2a compared to controls. Subsequent analysis of serum in CD4 mAb-treated animals revealed that mAb was present in the circulation for 14 days. Moreover, mice given CD4 mAb and dMTg, then challenged after only 10 days, when CD4 mAb was still circulating, developed a significantly higher incidence of thyroid damage than controls. These findings suggest that modulation of CD4 antigen does not interfere with Ts activation, but the presence of CD4 mAb, at the time of

autoantigenic challenge, can interfere with tolerance to EAT induction. Thus, the direct relationship between the presence of CD4 mAb and inhibition of EAT suppression implicates a role for CD4 molecules in the medication of suppression.

12/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09037705 BIOSIS NO.: 199497046075
Adhesion molecule monoclonal antibodies inhibit experimental autoimmune thyroiditis.
AUTHOR: Metcalfe R A; Tandon N; Tamatani T; Miyasaka M; Weetman A P(a)
AUTHOR ADDRESS: (a)Dep. Med., Univ. Sheffield, Clinical Sci. Centre, Northern General Hosp., Sheffield, S5 7AU**UK
JOURNAL: Immunology 80 (3):p493-497 1993
ISSN: 0019-2805
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To examine the role played by adhesion molecules in thyroid autoimmunity, we have assessed the effect of administering monoclonal antibodies (mAb) against intercellular adhesion molecule-1 (ICAM-1) and lymphocyte function-associated antigen-1 (LFA-1) in experimental autoimmune thyroiditis, induced by immunizing rats with thyroglobulin in complete Freund's adjuvant. The antibody against LFA-1, but not against ICAM-1, reduced thyroglobulin antibody production (P lt 0.01) and both antibodies caused a significant reduction (P lt 0.002) in the severity of the thyroidal lymphocytic infiltration. In vitro, both mAb impaired the proliferative response of splenic and lymph node T cells to thyroglobulin, but only the antibody against LFA-1 reduced thyroid cell killing assessed using splenic lymphocytes as effectors. Monoclonal antibodies against both these adhesion molecules appear to inhibit cell-mediated autoimmunity in vivo, but only the LFA-1 mAb reduced the autoantibody response.